IN THE UNITED STATES DISTRICT COURT

FOR THE EASTERN DISTRICT OF TEXAS

MARSHALL DIVISION

CENTOCOR, INC., ET AL.,	)(	
	)(	CIVIL DOCKET NO.
	)(	2:07-CV-139-TJW
VS.	)(	MARSHALL, TEXAS
	)(	
	)(	FEBRUARY 26, 2009
ABBOTT LABORATORIES	)(	9:00 A.M.

CLAIM CONSTRUCTION HEARING

BEFORE THE HONORABLE JUDGE JOHN WARD

UNITED STATES DISTRICT JUDGE

APPEARANCES:

FOR THE PLAINTIFF: (See Attorney Sign-In Sheet)

FOR THE DEFENDANT: (See Attorney Sign-In Sheet)

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(Proceedings recorded by mechanical stenography,

transcript produced on a CAT system.)

I N D E X

February 26, 2009

COURT SECURITY OFFICER: All rise.

THE COURT: Please be seated. Morning Counsel.

All right. We've got a claim construction hearing in 2:07-CV-139. That's Centocor, Incorporated, versus Abbott.

What says the plaintiff?

MR. SAYLES: May it please the Court, Dick Sayles for Centocor and New York University. We are ready. And with the Court's permission, may I introduce our team?

THE COURT: Okay.

MR. SAYLES: Seated at counsel table is Dianne Elderkin, who will be our lead today and will be presenting to the Court. This is Barbara Mullin.

MS. MULLIN: Good morning, Your Honor.

MR. SAYLES: And this is Matt Pearson. Each of these lawyers is from the Woodcock Washburn law firm. Mr. Ken Dow over in the corner is with Centocor. He is the vice president of intellectual property law. And, of course, Ms. Henson and Mr. Strachan from my office.

THE COURT: Okay. Thank you, Mr. Sayles.

Defendant Abbott Labs?

MR. RICHARDSON: Good morning, Your Honor, Michael Richardson. We are ready to proceed. With me

today is Bill Lee from Wilmer Hale.

MR. LEE: Good morning, Your Honor.

MR. RICHARDSON: He'll be taking the lead. Amy Wigmore and Bill McElwain. We also have here from Abbott Peter Witty and Eric Martin, and also Jamaica Szeliga.

THE COURT: Thank you, Counsel.

Mr. Richardson, tell Mr. Beck that I'm a little upset that he wasn't here. I wanted to have his views on chimeric antibodies today. I'm sure that he would have something valuable to say to all of us.

MR. RICHARDSON: He would have something very valuable.

THE COURT: Okay. Well, you tell him that I noted that for the record.

MR. RICHARDSON: He said to let you know that he'd be teeing off at about 9:30 today.

THE COURT: That's probably fortunate for all of us.

All right. We'll hear from -- we've got you an hour and a half per side, and so we'll hear from the plaintiff. The Court has read the patents twice. It's very interesting. I wish I understood. And I've been through, of course, your tutorials, and with staff, I've been through all the briefing with their doing most of

the heavy lifting on that. So it is a challenging patent for the Court.

MS. ELDERKIN: For all of us, Your Honor.

May it please the Court. Again, I'm Dianne Elderkin. I'm here to present the argument for the plaintiffs, Centocor and NYU. I'll be referring to them jointly as Centocor for the most part.

Of course, there are two patents involved in the lawsuit here. The '775 and '239 patent. They are co-owned by Centocor and by New York University, and the inventions disclosed in these patents relates to certain antibodies to a protein called tumor necrosis factor alpha or what we refer to in shorthand as TNF.

And as you'll hear as this case proceeds, Centocor and NYU contend that these patents cover Centocor's commercial product, Remicade, and also covers Abbott's commercial product, Humira.

I think it's fair to characterize both of these products as wonder drugs because each of these single antibodies can be used to treat a number of chronic debilitating diseases as varied as rheumatoid arthritis, Crohn's disease, which is a horribly debilitating disease of the gastrointestinal system, and psoriasis.

The common factor in each of these diseases

is that they result from an overproduction in the body of the protein TNF. TNF, of course, is a naturally occurring protein. It's involved in your immune system, but when it's overproduced, it can cause problems. The invention disclosed and claimed in the two patents-in-suit relate to a certain defined class of antibodies that bind to TNF in a way that causes it to lose its biological activity.

Now, just to step back for a minute about how this invention came about. It started with two researchers at NYU, Dr. Le and Dr. Vilcek, who worked to find a mouse or murine antibody that had this effect on TNF that could bind to TNF and neutralize its biological activities. And doing that is sort of like looking for a needle in a haystack because the body, whether it's human or mouse, can make thousands of antibodies to a particular target, but what they did is they finally found an antibody that did bind to TNF, bound strongly enough to TNF, bound to TNF in the right way that it would neutralize the activity of TNF. And the antibody that Dr. Vilcek and Dr. Le found, this mouse or murine antibody, is called A2.

Then the inventors at Centocor got involved to make a recombinant antibody based on that A2 mouse antibody. They used genetic engineering techniques to

make a chimeric antibody that was based on A2. So what they did was they found a way to replace a portion of the A2 antibody, the constant region, which is the -- you remember the Y structure of an antibody, the constant region is the base of the Y and starts up on the branches.

So the Centocor inventors found a way to replace the constant region of the A2 antibody with a portion of the antibody encoded by human DNA. So the resulting antibody is said to have a human constant region. And this genetically engineered body was called cA2, C for chimeric, and A2 because it's based on the original A2 murine antibodies. And cA2 is the preferred disclosed embodiment in the patents-in-suit. It is also the antibody that is in Centocor's commercial product, Remicade.

Now, as I will explain, what Abbott is attempting to do with its claim constructions is to limit the scope of the two patents-in-suit to a single embodiment, to the cA2 antibody, and that's the theme that runs through almost all of the claim construction disputes that the parties have.

I put up on the screen Claim 1 of the '775 patent. It contains many of the claim terms that are in dispute between the parties, but it also provides a nice

framework for just how are the antibodies of this invention claimed. Now, the inventors could have just claimed cA2 antibody, but they didn't. They claimed a group of antibodies based on the characteristics of the cA2 antibody. And as an example, in the claim here, some of the highlighted language is particularly relevant. The claim refers to an anti-TNF antibody. That's a term in dispute that we'll discuss.

It says that the antibody has a -- comprises a human constant region. That's a term that is not in dispute, and you'll recall that the cA2 antibody that I just discussed does have a fully human constant region. It is completely derived from human DNA, and there's no dispute between the parties that human in the case of the human constant region in the preferred embodiment is fully human or includes fully human.

And then the rest of the claim talks about how and in what way the claimed antibodies bind to TNF. It talks about the competitive inhibition, which we'll get to. It also says that this antibody must bind to a neutralizing epitope of the TNF and that it must have a certain affinity.

So these are the ways that the claims characterize the antibodies. Not all of the claims -- for example, the claims in the '239 patent don't include

that -- don't all include the recitation of affinity, but this claim fairly sets forth many of the terms in dispute.

The first term that I'll address is just what is the meaning of anti-TNF antibody? And there are three other terms that don't appear in Claim 1 which Abbott, in its briefing, grouped with its discussion of the anti-TNF antibody, and so we'll address them in that grouping, as well. And this slide, No. 6, shows all of those terms. Anti-TNF alpha antibody, but then these other terms that appear in some of the dependent claims, human variable region is, human light chain, and human heavy chain. And what we've shown in this slide is what Centocor's proposed constructions are for these terms.

For Anti-TNF alpha antibody, very simply that it is an immunological protein which, to be honest, is another word for antibody that binds to TNF alpha.

For the human variable region, the human light chain, and the human heavy chain, the parties don't dispute what a variable region, what a light chain is, what a heavy change is. The dispute centers around what does human mean?

Centocor's constructions require that the -- that human means that this particular region or chain are encoded by a gene derived from human DNA. And what

that means -- what encoded means is that the DNA -- the DNA has the instructions for making an antibody, and if the DNA which makes this particular antibody is derived from human DNA, then this region is encoded by a gene derived from human DNA.

Now, the common thread distinguishing Centocor's constructions of these terms from Abbott's constructions is that Abbott wants to insert limitations that would exclude fully human antibodies from either the discussion that the definition of anti-TNF antibody or would exclude the possible -- possibility of a fully human variable region, a fully human light chain, or a fully human heavy chain.

I can explain why that is not appropriate, but, first, there are a few places where there's some agreement. It's always nice to get to that. This slide shows the party's construction for anti-TNF antibody, and, obviously, both parties agree that this antibody needs to bind to TNF alpha, so there's no dispute about that.

In Centocor's construction, it's referred to as an immunological protein. Abbott refers to it as an antibody. In that regard, there is really no disagreement. Those things are pretty much interchangeable, but, of course, here's where the real

distinction is. Abbott with its construction of anti-TNF antibody is attempting to read extraneous limitations into the claim. It's attempting to limit this claim not to any type of antibody but only to murine, which is a mouse antibody, or chimeric, which it defines as an antibody that has DNA sequences from different species.

And if you recall, this is a screen shot from Abbott's tutorial. If you recall, they talked about the different types of antibodies. There is a fully mouse antibody. That was -- that's what they depicted in green, a murine antibody, a type of chimeric antibody, such as cA2. Centocor's preferred embodiment is shown in the middle in the top row here, and there there is a constant region. The human region is -- I'm sorry, the constant region is human derived from human DNA. The variable regions are shown in green, and they are still from a different species, from mouse. The next antibody on the top right is what is frequently called a humanized antibody. There it has a human constant region. The variable region is mostly human, but the very tip of the variable region, the region that binds to the antigen, also called the CDR, is mouse, and then at the bottom you have a fully human antibody.

So there's no dispute that these are all

antibodies. The dispute is that Abbott wants to define the term antibody in the claim as excluding the bottom one there, the fully human derived antibody.

Now, again, since I said that the constructions of these other terms are all interwoven with the anti-TNF antibody, let me quickly look at the party's different constructions for these other terms.

Human variable region, again, there's no dispute between the parties as to what a variable region is. This is a diagram for Centocor's tutorial. The variable region is shown in blue, the constant region in green, and the different depictions here -- the stick figure is sort of an easy depiction, but a more realistic slightly more three dementia figure of what an antibody actually looks like is shown in the large figure on the right.

So there's no dispute between the parties about what a variable region is. The dispute here is that Abbott says -- I'm sorry, let me go -- this is a screen shot from Abbott's tutorial. And, again, the same thing, they don't dispute that the variable region is the top part of the Y going all the way out to the end of the Y, and the constant region is below that.

The dispute between parties in this construction, again, is that Abbott wants the human

variable region to actually have a nonhuman portion, that the CDR, the binding region, must be grafted from a nonhuman species. You recall that the CDR, as depicted in this figure from our tutorial, again, is just the small part at the end of the variable region, and that's the important region which binds to the antigen or the TNF.

Much the same dispute exists with respect to the construction of human light chain and human heavy chain. For both of these, there's no dispute between the parties what human -- I mean, what heavy chain and light chain mean. As depicted in our tutorial, the heavy chain is the portion of the antibody that forms a Y structure. The light chain are two chains that are attached to the branches of the top of the heavy chain.

The only dispute between the parties is whether the heavy chain and the light chain must also have some nonhuman portions, even though the claim refers to human heavy chain and human-human light chain.

So why are Abbott's attempts to incorporate extraneous limitations wrong? Well, first of all, it's improper to limit the plain and ordinary meaning of the claim language absent a clear intent to limit scope. The claim refers to an antibody. It doesn't limit it to a murine antibody or a chimeric antibody. The claim

refers to heavy constant regions -- I mean, to heavy light -- heavy light -- sorry. Human light chain, human heavy chain, and human variable regions. It doesn't say partly human. So the plain language is very clear. So there has to be some very clear intentions to limit the scope, and that's very clear from the case law that's cited in our brief.

And the cases that Abbott cites really are not on point. The SciMed case that they cite makes it clear there has to be a very strong indication of disavowal to depart from the plain meaning of the language. In the SciMed case, it was a medical instrument that involved two lumens or tubes, and the issue is whether the tubes could be coaxial, one within the other, or whether they had to be side-by-side in the instrument.

And the Court -- the Federal Circuit said that the Court had properly construed the claims as limited to coaxial because the patent described the invention and all embodiments of the invention as having a coaxial lumen. This Court found that was the kind of plain and clear disavowal that would allow you to read in limitations to the claim.

Another case cited by Abbott that is also not instructive for our fact situation is the

Astrazeneca case. In Astrazeneca, the general summary or description in the patent described a feature of the invention and criticized other products that lacked that same feature. So that operated as a clear disavowal. There is no clear disavowal here.

The best disclaimer or evidence -- purported evidence of a disclaimer that Abbott can find that's referenced in its brief is a statement that was made in the first patent application that Centocor filed in 1991, which doesn't even appear in the patent anymore. And that is a statement that was made in the originally filed application, the '827 application, but subsequently removed, and all that statement does is note that a certain existing method for producing human antibodies had some drawbacks. But the '827 specification also described a solution to that problem using certain recombinant DNA technology to isolate an antibody gene from a human B cell, and that's at Exhibit 12 at 23.

Further the '827 patent also describes potential advantages of using human antibodies instead of mouse antibodies in terms of greater utility for treating chronic conditions. So this is far from the kind of unequivocal statement that the cases like SciMed and Astrazeneca need for disclaimer.

It's also apparent that there's no disclaimer from the fact that the specification of the patents actually discloses human antibodies, and here on Slide 20 we show an excerpt from Column 5 of the patent where it says, Anti-TNF antibodies are intended to include a number of antibodies, and it mentions human antibodies.

Abbott has cited no case where the Federal Circuit has said it's okay to insert a narrowing limitation where the patent itself says that something is included. Further, the patent discloses human regions. This is an excerpt from Column 12 of the patent, and it talks about technique used to raise antibodies of the present invention, and it says such antibodies include human-human antibodies.

Now, what -- what Abbott is trying to argue that when the patent refers to human-human antibodies, generally, with this hyphenated nomenclature here like murine-human, it's the murine variable region and a human constant region. So what Abbott is trying to say is that when there's a reference human, hyphen, human, the first human really means, well, not all human because it has a mouse CDR region.

The problem with that is it's internally inconsistent. If the second part of the human in the

human, slash, human we know includes fully human as the fully human constant region in the preferred embodiment, how does it make sense, then, to construe human before the hyphen to mean something different? The answer is it doesn't make sense.

THE COURT: What do you say, though, about where you define -- where the patent defines chimeric as being from a different species, and then later on in the patent, it says -- it talks about a chimeric antibody including a human-human?

MS. ELDERKIN: The patent is not entirely consistent in that regard, and we admit that.

THE COURT: I think that's a fair statement that it's not consistent. You think I can just disregard that inconsistency or what?

MS. ELDERKIN: No, no, we contend that when someone skilled in the art reads the patent in its entirety, that it would be apparent that the inventors intended to include and that the patent describes fully human antibodies. Let me -- let me find the right disclosure of that to point to you.

THE COURT: Well, chimeric is defined at the bottom of Column 10, it looks to me about Line 64. And then -- then I find conflicts over at Column 20 at about Line 45.

MS. ELDERKIN: May I go over and get my copy?

THE COURT: Absolutely. I have a habit of not trying to mark my patent up. Whatever column you throw up on the screen, make sure that I have at least marked that as something I ought to be looking at. So I wanted to ask you about those.

MS. ELDERKIN: And, I'm sorry, Your Honor, you're looking at the bottom of Column 10.

THE COURT: Yeah. It says --

MS. ELDERKIN: Right.

THE COURT: That's where it defines in the patent chimeric, correct?

MS. ELDERKIN: Well, there is -- there is a reference to chimeric antibodies there. We will point out --

THE COURT: You don't think that's an express definition of chimeric?

MS. ELDERKIN: Actually, we would refer to the top of Column 14.

THE COURT: Column 14, all right. Let me get there. Just a minute.

MS. ELDERKIN: Okay.

THE COURT: Okay. There. That's right.

MS. ELDERKIN: And there the term is

actually used in quotes, as is the case in other parts of the patent where definitions are being provided, and at the top of Column 14 --

THE COURT: Well, it says it includes monovalent, divalent, and polyvalent, but that doesn't seem that it's really defining it there. That's a characteristic. It can include any one of those three bondings, correct?

MS. ELDERKIN: I'm sorry, Your Honor. I'm trying to find the...

THE COURT: Well, I don't want to take up too much of your time. You told me -- you acknowledged to me that there are some inconsistencies, and I'm trying to determine how I'm going to handle that or how you would suggest I'm going to -- what I'm going to say that's going to end up in the Federal Circuit and pass muster, you see. That's what I'm -- I get down to sort of that level.

How would you -- what would you think would explain this inconsistency to the extent that I could adopt your position? That's what -- I mean, that's really what I'm asking you.

MS. ELDERKIN: Well, I think there are several things, and I will -- I see that on my next slide I get to the section I was looking for. So I'm

sorry for the disarray here.

First of all, we would argue -- of course, the term chimeric is not a claim term, so it's not in the claim. The claim talks about human regions, human chains, and antibodies. So whether or not a chimeric antibody includes mouse parts -- must include mouse parts are not is an interesting issue, but we're not really defining what the term chimeric means. We're defining what the plain meaning of the terms in the claim are and whether there is a clear disavowal based on what's in the specification.

So even though we would grant that there are statements in the patent that, frankly, we wish were not there, there are other statements that support our position that a chimeric antibody can include an antibody that's completely from human sources but from two different human sources. The chimeric really requires that you have two different sources of the DNA. It's not necessary that they be from two different species.

And an example of that is -- support for that is what I show here on Slide 22. This is a statement from Column 14 -- bottom of Column 14 to the top of Column 15 of the patent.

THE COURT: Just a minute. Let me make sure

I've got that marked.

MS. ELDERKIN: Sure.

THE COURT: Okay. It goes from 14, 64.

MS. ELDERKIN: Right. To Column 15, Line 1. What this is saying is basically disclosing how you could make human constant -- human CDR as binding regions that would be in the variable region of the antibody. It says that the antibody producing cell contributing the nucleotide sequences encoding the antigen binding region, again, that's that CDR region, can be produced by transformation of a nonhuman cell or a human cell.

So this is saying that you can get those binding regions. The CDR region, which Abbott is saying, oh, it has to be mouse, it has to be nonhuman. This is saying, well, you can get it from a human cell.

Now, Abbott, we suspect, based on something its expert said, may argue that you can contort the reading of this to mean that, well, you would take mouse DNA and put it into the human cell, but their -- their expert never explained why one would go through that kind of convoluted procedure to obtain a CDR region.

So we contend that this Column 14, which was in the very first patent application filed, this statement, is support that one reading the patent with

everything that's in there, good and bad, would look at this and understand that, well, this is covering fully human -- the claims have to encompass fully human variable regions and fully human CDR regions.

THE COURT: Thank you.

MS. ELDERKIN: Another thing that is telling here is that Centocor could have claimed partially human if it had intended to do so. There is reference in the patent, for example, Column 6, Lines 27-32, to chains that have at least part human constant and at least part of a variable region of nonhuman origin. So it could have claimed partly human if that's what it intended to do, but it didn't use that language. It used the broader language, human, which it broadly, as we construe it, means partially -- it means derived from human DNA.

Now, Centocor -- Abbott in its brief also pointed at 18 -- Page 18 of its brief, also pointed to language in this specification that it says defines the invention in places as limited to chimeric and murine antibodies. Again, we had the dispute over whether chimeric must mean at least partially nonhuman. We disagree that it does. But even if it did, we'd have two other points in response to those arguments.

First of all, the statements in the

specification that they refer to are not restrictive. The patent does disclose murine and murine-human antibodies, and those passages can be read simply as referring to those particular embodiments. And the Anderson case that Abbott cites, I believe on Page 18, as support for limiting the claims to the particular embodiments disclosed, again, is not on point. In Anderson, the claims were limited to certain features because the patent specification required -- made it clear that those features were required, and that, again, is not the case here.

So to try to summarize some of the reasons why Abbott's attempt to incorporate extraneous limitations into the claim is wrong, it's improper under the law to limit the plain and ordinary meaning of the claim terms absent -- absent clear intention to limit the scope. And there's no evidence that there was such a clear intention -- a clear disavowal here.

Abbott's attempt to limit the recitation of human when it refers to variable region or light chain or heavy chain is inconsistent with the recitation of human as defining the constant region of the claim. Again, there's no dispute between the parties that a human constant region can encompass a fully human constant region. So where the word human is used in

Claim 1 to refer to the constant region, no dispute, it can be fully human.

On what basis, then, can Abbott say that the term human, when it's used in Claim 2 or 3 to refer to the variable region or light chains or heavy chains has to mean something else? When a term is used -- terms are generally used consistently in claims, and there's no good reason not to do so here.

Abbott's construction would exclude disclosed embodiments. We think maybe they backed away from this position. Their construction would exclude murine antibodies. I think that's right. No, I'm sorry, their construction would -- I'm sorry, yes, it would exclude disclosed embodiments because, as I just explained, there are embodiments disclosed or at least mechanisms disclosed for making antibodies that have human CDR regions.

And, finally, Abbott improperly seeks to raise new matter issues. They discuss in their brief when certain points in statements in the specification were added, and saying, well, because this wasn't in the original application and wasn't added until such and such a date, you can't rely on it. I suspect that's an argument we may have at a later date, but that's not an issue here on --

THE COURT: I anticipate this will not be the last time that I hear this.

MS. ELDERKIN: It's not an issue, though, Your Honor, for claim construction.

THE COURT: I agree with you about that.

MS. ELDERKIN: Okay. Then I'll say nothing more on that.

THE COURT: I don't think it's an issue for today, but it is obviously an issue.

MS. ELDERKIN: Great. So, in summary, Centocor's claim constructions, we think, are the appropriate ones, that they are consistent with the use of the terms in the specification with the plain meaning, no extraneous limitations being brought in, and we would ask the Court to adopt those constructions for the terms anti-TNF antibody, human variable region, human light chain, and human heavy chain.

The next term I'd like to discuss, and there's really not much dispute on this, but it's sort of a predicate for discussing competitive inhibition where there is a dispute, and that is the term binds to a neutralizing epitope. What is a neutralizing epitope?

And, again, there's very little daylight between the parties' constructions of this term. We both agree that there's some aspect of binding to TNF in

a way that there's a loss of biological activity. The real difference is that Centocor's construction defines it as a noun. Abbott's construction is more functional. It's an important distinction only because the fact that the patent does recite that there's binding to a neutralizing epitope is going to be important when it gets to discussing the next term, competitive inhibition. It's important that the patent does say the antibody that's covered by this claim has to bind to -- has to bind to TNF. It has to bind to a neutralizing epitope. So that's all I'll say about that.

I'll turn, then, to competitive inhibition because there is substantial disagreement between the parties on the construction of this term. So, again, this -- what the claim language is is competitively inhibits binding of A2, and A2, again, is the mouse antibody, to human TNF alpha. And there's a parenthetical expression there after A2, and it says, ATCC Accession No. PTA-7045. And what that is, it's a reference to the cell line that expresses or makes the A2 antibody that's been deposited with a nonprofit organization, so it's available to the public. So there's no dispute between the parties over what that means.

Centocor's construction quite simply is,

well, competitively inhibits just means that competes, that whatever antibodies covered by this claim has to compete with A2 for binding to human TNF alpha. And the patent discloses a method for determining competitive inhibition. This is in Column 12 at Line 16 to 23. And the patent says, preferred methods for determining antibody specificity and affinity by competitive inhibition can be found in Harlow, the Harlow manual, a very established lab manual, which is actually incorporated by reference into the patent.

So Harlow describes an affinity -- a competitive inhibition test, and just that kind of test was carried out for the A2 antibody, and it was disclosed in the patent in Example X10 and in Figures 9A and 9B. And you might recall this figure from the tutorial that Centocor submitted talking about competitive inhibition.

This is a somewhat simplified version of the graph that's shown in Figure 9A of the patent, and what's happening here is TNF is the target. It's the antigen. What you do is you put that in a plate, it's adhered to a plate. The particular test in Figure 9A was carried out to determine if A2, the mouse antibody, competes with cA2, the chimeric antibody based on A2.

So what happens is in the first test, they

took A2, the mouse antibody, and they labeled it so that it could be detected, and they add it to the plate that TNF is bound to, and they allowed time to bind to the TNF, if it's going to do so, and then they wash off what's left, and then they measure -- because that A2 is labeled, they can measure how much of the A2 is bound there to the TNF. And that's the first point that you see on this graph on the Y axis. So only A2 is added, and there's 1.4 units of antibody bound to the target, so a lot of antibody bound to the target.

So then what they do is they take a mixture of A2, and they add some of the cA2 to it, and they apply that to a plate, allow time to bind, and wash it off, and then we see, well, how much A2 is bound now? And if it's less bound than was bound when there was no cA2 there, that suggests the two antibodies are competing. And they run -- they continue the test and run a series of tests with additional amounts of the test antibody, cA2, added each time to see what happens.

You get a curve in this particular case that looks like this. This is not an uncommon curve for a competition -- a competitive inhibition assay, and the experts would say this shows two antibodies compete with one another.

So Centocor's construction is that

competitive inhibition really means that the antibody competes with A2 for binding to TNF alpha and that this could be measured using a standard assay such as that described in the patent.

Abbott's proposed construction, I presented on this slide, Slide 32, and it's quite a mouthful. I can't get it all on a slide along with our construction. It's quite complicated, but let's try to break it down. The first part of Abbott's construction, there really is no dispute, the part I've highlighted in red. We agree with the ATCC accession number means and then a product of that self -- self line is the A2 antibody which binds to human TNF alpha.

We agree that an antibody competitively inhibits. One way to determine that is using a standard ELISA or equivalent assay. And if you recall from Abbott's tutorial, they describe a test much like I just described with the Harlow test where you have TNF bound to the plate and you add increasing amounts of the test antibody and see what happens to the binding. And they refer to that in their slide as a competitive inhibition experiment. So there's no dispute between the parties that that is a test for determining competitive inhibition.

At the bottom of this slide or the bottom of

the end of their construction, Abbott includes a definition of epitope, and we would merely note that that definition is inconsistent with the definition of epitope in the patent. It's not the same as the definition in the patent, which appears at Column 13, Lines 15 to 17, which says that the term epitope, in quotes, is meant to refer to that portion of any molecule capable of being recognized by and bound by an antibody at one or more of the antibodies' binding regions, so there is some difference in that.

But the real meat of the dispute has to do with Abbott's attempt to read in extraneous limitations. They want to read in a limitation that the antibody, to meet this competitive inhibition requirement, must bind to the very same epitope as the reference A2 antibody and also some quantitative limitations that it must bind -- it must compete as strongly for binding to A2 as does the A2 with itself. And we contend that there is no basis for doing this.

First of all, we contend Abbott is improperly trying to read in the term same epitope into this claim limitation. For one thing, the claim already defines the epitope to which the antibody binds. As I pointed out earlier, the claim says it binds to a neutralizing epitope of TNF. So if the claims have to

bind -- if the claimed antibodies have to bind to the same epitope as A2, that makes this language, neutralizing epitope, superfluous and unnecessarily because we know that A2 binds to a neutralizing epitope.

Further, none of the evidence that's cited by Abbott supports this same epitope limitation. So let's look briefly at what Abbott's relying upon. First of all, it's somewhat striking that Abbott's own expert admitted that Abbott's construction requiring same -- same epitope binding is not consistent with the plain meaning. He was -- Dr. Marks was asked: So setting aside the patent specification here, in your opinion, someone skilled in the art would define the term competitive inhibition to require binding to exactly the same epitope? He said, No, not exactly the same epitope. Outside the specifications. No.

So the plain meaning clearly does not require what Abbott is trying to introduce into this claim construction. So is there something in the patent that requires it? Quite the contrary, the patent actually distinguishes between competitive inhibition and identical epitope. Here on Slide 39, we have an excerpt from the patent at Column 12, Lines 4 to 15.

There are two classes of preferred antibodies disclosed in this paragraph. And the first

said, There are preferred anti-TNF antibodies, and those are those which will competitively inhibit binding to human TNF alpha of the mouse antibody A2. And that's more or less paraphrasing what's in the claim. Then it says, well, there are -- other preferred antibodies are those that bind the epitopes recognized by A2. So it talks about them not as being coextensive. It mentions them each as separate and distinct possibilities. So that does not support Abbott's suggestion that the claim requires that the antibody bind to the same epitope as A2.

Further, the Harlow manual, this is the manual that discloses the competition assay, it's incorporated by reference. It talks about, well, if you get this competition what it could mean. It says, well, if the sites of the interaction of the two antibodies are identical or overlapping, the unlabeled antibody will compete. So Harlow says -- acknowledges it doesn't have to be the same epitope, it could be overlapping, and it could be other things, as well, as you will hear.

Abbott's expert looked at a statement made in the prosecution history to the effect that the claimed antibodies must bind to the same or similar antibody -- epitopes and concluded that same or similar means the same. I think I need to say nothing further

about that.

So there is no support in the specification for reading in the same epitope limitation. Let's look at Abbott's attempt to read in a quantitative limitation, a requirement that the antibody being covered must compete with A2 as strongly as A2 competes with itself.

The patent is silent about any kind of quantitative limitation on the competition to begin with. Abbott has made a suggestion, well, the claim would be invalid and indefinite if you don't put some limitations on it. We contend that invalidity is not an issue here today on claim construction, but the outer limits of the claim don't have to be precisely defined. It's well established in case law that that's the case.

It will be an issue of fact for the jury based on what they hear at trial to determine whether the Humira antibody competes with A2 providing two TNF. The Harlow reference, again, that lab manual, actually has a section that says, well, if you want, you can make this competition -- competitive inhibition assay quantitative. It's an option. It's not something that you have to do. That's at Exhibit 17 at Page 1725901, again, indicating to those skilled in the art -- indicating that people skilled in the art would

not understand that a competitive inhibition assay must be quantitative in order for it to be meaningful.

And, importantly, what Abbott's trying to do here with its same epitope and same level of inhibition construction is to effectively limit the claim scope to a single cA2 antibody. If it has to bind to the very same antigen -- epitope and it has to bind the very same way that A2 does and A2 has the same binding region as cA2, in essence, they are limiting this claim to the single disclosed embodiment which is improper under the law, especially under the circumstances here.

A few arguments that Abbott made in its brief that clearly invite error. At Page 21, they say the specification's only example of competitive inhibition is testing between A2 and cA2. These antibodies bind to the same epitope. Well, fine, but there's nothing in the law that allows the Court to limit the claims to just the examples in the patent, absent extraordinary circumstances such as a disavowal.

Another argument that they make at Page 23, that the specification does not describe or even suggest any examples of antibodies that inhibit one another by any method other than binding to the same epitope. Again, we don't have to have claim scope coextensive with the scope of the examples in the patent. And the

last part -- point here is pretty much the same thing. They say inhibits should be construed to require a level of inhibition that is the same as the inhibition exhibited by A2 against itself. Again, there's no basis for limiting these claims to preferred embodiments.

So for all these reasons, Centocor contends that its construction, which very simply, in accordance with the plain and ordinary meaning of the term, construes the competitively inhibits clause to mean competes with A2 for binding to human TNF alpha.

With that, let me turn to the subpart two of Claim 1, which is a reference to the affinity with which the antibody binds to TNF. An affinity, of course, has to do with the strength of the binding to the antibody -- of the antibody to the target TNF, how much energy is required to get the antibody on there and how much energy is required to pull it off.

The parties essentially agree on part of this construction, as I said before, in the binds to a neutralizing epitope. This is where we have a minor disagreement about whether it's a noun or should it be described functionally, but we depart on how affinity is measured. The language of the claim -- let me go -- here's the claim language. It says, binds to a neutralizing epitope of human TNF alpha in vivo with an

affinity measured as an association constant as determined by Scatchard analysis.

And where the parties disagree is that Abbott says that you have to use that Scatchard analysis to measure the affinity in the living organism. Centocor's construction, of course, doesn't require that.

So let's talk a little bit about the Scatchard analysis so you can understand why Abbott's construction really doesn't make sense. The Scatchard analysis as described in the patent can involve labeling the antibody with radioactive material and then measuring affinity. To carry out the Scatchard analysis in the human body, in the organism, as Abbott contends, you would have to insert radioactive material into the body. That simply isn't done. In fact, Abbott's expert admitted that he wasn't aware of a single instance where a person was ever injected with a radio-labeled antibody to measure affinity.

It's also undisputed that the Scatchard result that can be carried out in the lab, not in the body, correlates to the affinity with which the antibody will bind in vivo. That was like many tests that scientists do in the lab to try to determine and ascertain, well, what kind of activity will this

antibody have in the body?

So where the claim refers to binds to a neutralizing epitope in vivo, we know that means the antibody binds to TNF in our bodies, and that we measure the affinity using this Scatchard analysis, and that that will correlate to the strength with which the antibody binds in the body.

This is another Abbott construction that we contend invites error. First of all, it's contrary to the plain meaning to the skilled artisan and it's nonsensical to suggest that this would be done with radio-labeled compounds in the body. It would also exclude the disclosed embodiment from the scope of the claims because the disclosed embodiment, the cA2 antibody, its affinity was measured using a Scatchard analysis in a lab, not in a body. That's the only measurement that was made. So there's simply no reason to adopt Abbott's construction.

They cite the Chef America case, which really is not on point here. The Chef America case, as you remember, there was claim language that required that some -- dough is a baking thing -- that some dough be heated to 400 to 800 degrees. And the patentee said, well, you know, we really didn't mean to. We really meant it should be heated at 400 to 800 degrees. And

the Court said, I'm sorry, the only reasonable construction of this claim, based on the language that you've used, is that it's heated to 400 to 800 degrees, so we're not going to rewrite your claim for you.

But here we're not asking anybody to rewrite the claim. There is a reasonable construction of the claim that Centocor has proposed that's consistent with the language of the claim and how people skilled in the art would understand it. So the Chef America case is really not on point and does not compel Abbott's construction.

So to summarize the -- Centocor's construction does not require carrying out the Scatchard experiment in the body, and our construction is otherwise rather straightforward.

Now, we get on to some of the terms where I think there are some less disputes, and we can probably move somewhat quickly. Recombinant, Claim 1 refers to this as a recombinant antibody, and what does that mean? Centocor's construction says that this means that it's encoded by DNA made with recombinant DNA technology, for example, encoded by a gene that was built by splicing DNA. So both parties agree that a recombinant antibody is one that's made by manipulating or splicing DNA. There's no difference between us there.

But Abbott then inserts an element of indefiniteness in its construction by adding not substantially by natural immunization techniques. And we've asked them what that means, and we really haven't gotten an answer to that, so we are troubled by the idea of putting something in the claim that would make it indefinite.

The problem with this is that an antibody such as cA2, which is an embodiment of the patent, did start with immunizing a mouse. It was the mouse antibody, A2, that started the whole process that led to the chimeric antibody, cA2, was made by immunizing a mouse and then collecting the antibodies and finding the A2 antibody. So we are concerned that the language not substantially by natural immunization techniques adds a level of uncertainty that is not called for, not necessary, and will only cause problems.

Another term that the parties dispute is specificity. This is a term that's in Claim 9 of the '775 patent. It says that the antibody has specificity for a neutralizing epitope of human TNF alpha. So why do we care about specificity? Well, we want this antibody, this therapeutic antibody to go in and bind to the thing we care about, TNF, and hopefully not bind to something that's going to cause a problem for us.

So how do we define, then -- well, how do we determine whether it's specific or not? Centocor contends that specificity is clear -- it's clearly defined in the patent as referring to the fact that the antibody binds to TNF alpha but not to TNF beta. Abbott is inserting a species specificity aspect to the definition instead. But let me show you why Centocor's construction is correct here.

The patent describes specificity in terms of TNF beta quite clearly. At Column 49, Lines 29 to 41, it says the specificity of cA2, which is the preferred embodiment, for TNF was confirmed by testing for cross neutralization of human lymphotoxin, which is TNF beta, and then when you get to the bottom of that paragraph, it says, the results indicated that the antibody was ineffective in inhibiting or neutralizing this human lymphotoxin confirming the TNF alpha specificity of the chimeric antibody.

There's another reference to specificity in terms of whether or not the antibody binds to TNF beta or not at Column 21, Lines 14 to 16, and that says much the same thing as what I've shown here from Column 49.

THE COURT: Well, also, you -- you're citing from that Example 10, aren't you, started on Page -- Column 48? Isn't that where that came from in the

patent?

MS. ELDERKIN: Column 48, Example 10.

THE COURT: I mean, the Example 10 starts on Column 48.

MS. ELDERKIN: Yes.

THE COURT: It goes over in -- part of that example is what you have cited to me over on Column 49, Line 42 to 50, right?

MS. ELDERKIN: Yes, exactly, that's part of Example 10. That's the reference --

THE COURT: Doesn't that same -- doesn't that same example talk about this limitation that they're proposing?

MS. ELDERKIN: The species specificity?

THE COURT: Yes.

MS. ELDERKIN: That's hard for me to say, so I'm going to blow that. Yes, and, actually, that's further down in the same column.

THE COURT: That's what I'm saying, it's in the same example. I just wondered why it -- yours is correct and theirs -- I'm not saying theirs is correct, but I'm wondering why both of them aren't -- shouldn't be included in the limitation since they're found in the same example?

MS. ELDERKIN: Right. Well, where they're

talking -- where the -- of course, the patent language is -- the language in the claim is specificity, not species, but specificity, and the patent at Column 49, the portion I just referred to, says --

THE COURT: But the title -- the title of Example 10 on Column 48 is specificity of an anti-TNF, chimeric antibody, and I'm just saying that both of those examples -- within that example, you've got the discussion of two different -- two different times about specificity, it seems to me, and I'm asking -- what I'm trying to find out is why you believe I should -- I should include this but not include their species.

MS. ELDERKIN: Because this, what I'm referring to is Column 49 starting at Line 29, talks about specificity of the antibody, and that's the language in the claim, specificity. Down further in the column at the bottom of Line 49, and I have this up on Slide 57, it talks about the species specificity. Actually, I guess this is not on my slide here, but at the bottom of Column 49, it says, therefore, cA2 appears to share species specificity with the antibody A2.

So there is a distinction between specificity, and where the -- this example talks about specificity, it's talking about specificity with respect to TNF beta, but then down below, talking about species

specificity, and since the claim does not say species specificity, it just says specificity, the construction relevant to TNF beta we contend is the appropriate one.

THE COURT: You just want me to ignore the title that the patentee gave to the entire discussion?

MS. ELDERKIN: I think the term is used -- is used generally there because this example talks about a number of things, but the -- the explanation of specificity in the example breaks it down to specificity and species specificity, and we contend that the TNF beta example is the appropriate one.

THE COURT: Okay.

MS. ELDERKIN: That's also the example that has therapeutic meaning. If we're making an antibody to TNF that they want to put in your body to treat rheumatoid arthritis, we don't really care if it's going to bind to monkey TNF or chimpanzee TNF. We care if it's going to bind to other proteins in your body that might have -- where it might have a bad effect. Maybe it will turn on something good that you want to happen in your body.

So from a therapeutic standpoint, what really matters is whether the antibody is specific to the proteins in your body, and that relevant connection here is -- TNF beta is one of the proteins that's most

closely aligned to TNF alpha. So if the antibody is not binding to that, it indicates that it's likely not to bind to any of the other proteins in your body, as well.

I'm sorry if I spoke over your question.

THE COURT: No, no, I was just going to say, I didn't mean to cut you off either. I was just going to say you want me to say that I'm going to look at this strictly what is most therapeutic? I mean, it seems that would be your argument there, that I should be looking only at this patent at the specificity as to what's most therapeutic. I mean, that's not exactly what -- I'm just trying to figure out why you want -- think it would be appropriate for me to ignore this example -- part of the example. That's what I'm asking you.

MS. ELDERKIN: I think I would just repeating my answer.

THE COURT: Well, I think you would. Okay.

MS. ELDERKIN: Okay.

THE COURT: Thank you. Go ahead.

MS. ELDERKIN: So, again, we contend that the proper definition has to do with specificity of the TNF beta and not to other species.

Claim 11 of the '239 patent talks about inhibiting that -- the antibody inhibiting a

pathological activity of human TNF alpha, and, again, there's not a lot of difference between the parties here.

Centocor believes that since -- that you can't ignore the term pathology. It means something different than biological activity because other claims talk about biological activity, and that pathological means associated with a clinical problem.

I think we're pretty much on the same -- at the same place as Abbott. Our one concern is the last two words of their construction where they say that this activity must be associated with a disease or damage, and we're -- we're unclear about what damage means and don't want any indefiniteness incorporated into the claim construction.

'239 patent, Claim 14, talks about the anti-TNF antibody being produced recombinantly. This is another construction where we submit that Abbott is improperly trying to incorporate extraneous limitations. Our construction is simply that produced recombinantly means produced in a recombinant host cell, in other words, produced from a source, an organism or a cell line that includes a gene that was built by splicing DNA.

Remember there's recombinant technology

involved, taking pieces of DNA that don't naturally occur together and splicing them together to form new instructions for making an antibody.

The problem that we see with Abbott's construction is that we don't understand what the last four lines or so of it means. They talk about altering the genotype and phenotype of the cell, and we don't understand what that means. And then they also require that the inserted DNA be replicated along with the natural DNA. There's no basis for making that requirement. In fact, there is a reference in our patent to a method where the host DNA is not replicated along with the artificially-introduced DNA. That's at Column 30, Lines 23 to 25, and Dr. Marks also testified about that in Exhibit 9 at 165, Lines 8 to 18. So we think it's improper and unnecessary to incorporate this additional language which is not even that clear into this otherwise straightforward claim construction.

And with that, Your Honor, I have completed my presentation. I would be happy to answer any questions or --

THE COURT: On this word of recombinant and produced recombinantly, I mean, is there -- maybe this Court's a little bit simple, but has this got anything to do with -- this produced recombinantly, does this

have anything to do with produced in large quantities? Is that an issue at all?

MS. ELDERKIN: No. We don't believe that it does, Your Honor. It just refers to being made in a recombinant host cell as opposed to being made by chemical synthesis or by using a hybridoma.

For example, I think this appears in Claim 14 of the '239 patent, and the next two claims in that patent, one -- Claim 15 and 16, say rather than being produced recombinantly, they say, well, produced by chemical synthesis or produced by using a hybridoma. So I think that the language is just meant to, for one thing, distinguish from those types of synthesis, but it doesn't really go to quantity or ease of manufacturing.

THE COURT: All right. I don't have any other questions. Thank you.

MS. ELDERKIN: Thank you.

THE COURT: Mr. Lee?

MR. LEE: It will take half a minute, Your Honor, to change --

THE COURT: I understand. Mr. Lee, if you have a point during your presentation that you think is a natural breaking point, the Court would --

MR. LEE: How about now, and we could get it hooked up? Is that all right?

THE COURT: Well, that will -- that would probably be as good idea as any. Why don't we take about a 15-minute break?

MR. LEE: Thank you, Your Honor.

COURT SECURITY OFFICER: All rise.

(Break taken.)

COURT SECURITY OFFICER: All rise.

THE COURT: Please be seated, except for you, Mr. Lee.

MR. LEE: Thank you, Your Honor.

THE COURT: All right. Let's proceed.

MR. LEE: Your Honor, Centocor began its presentation today by describing the story of the invention, which in most claim construction hearings it's not precisely relevant, but in this case, it actually is and this is an issue on which we agree.

There was one claimed invention which was this chimeric antibody cA2. It was the only antibody invention. It was done in 1990 to 1991, and then, as Your Honor knows, there have been a series of 14 or 15 applications that had followed that result in some of the confusion that Ms. Elderkin and you discussed.

I think to answer one question Your Honor asked Ms. Elderkin or at least to give you our answer, there are inconsistencies and contradictions in the

specification. There are particularly inconsistencies if you give the incorporation by reference statements, and I'll come to that very shortly, their full meaning.

I think the answer to the question that Your Honor asked is in Section 112, which is the patent has a public notice function to tell you and to tell the public what's claimed, and those contradictions, to the extent they exist, ought to be resolved against the person who has the power of the pen, which is the patentee, rather than the public.

Now, there are 10 disputed terms and phrases in the Markman hearing, but there are really sort of two fundamental issues, and I'm going to devote the vast majority of my time to those, very quickly hit on three others and two cases, I think, suggesting a resolution that would actually leave less for the Court to resolve. But I'm going to focus on the two issues because I think that resolves most of the claim terms in dispute.

And those two issues are whether the claim terms anti-TNF alpha antibody, human variable region, human light chain, and human heavy chain cover fully human antibodies and their components. And then, secondly, the competitively inhibits portion of the claim.

What's on the screen now, Your Honor, on

Slide 3 of our presentation is simply the competing claim interpretations which Ms. Elderkin put on separate slides but accurately stated, and I think has fairly characterized the dispute, although we suggest it should be resolved in a different way.

On Slide 4, which I'll pass quickly, is just a refresher on the material that was in our tutorial on heavy chains, light chains, variable domains, and the complimentary determining region. The one thing I would say is if I focused Your Honor on the circle in the upper right-hand corner of the variable domain, the variable domain has two parts. There's a framework region, and there's a complimentary determining region, and that becomes important when we look carefully at the portions of the specification upon which Centocor relies.

Now, with a little color added to the different types of antibodies, well, maybe a little humor added to the different types of antibodies, there's really no dispute between us that there are four different types of antibodies developed in different ways at different points in time that are relevant to the Markman determination.

The first on the left are mouse antibodies that have been made by infecting a mouse with an

antigen, harvesting the mouse spleen, and isolating the B cells. The second, the chimeric antibody, which is the cA2, which Centocor claims to have invented, is something made using recombinant techniques that has a mouse variable region but a human constant region. The third category is humanized, and it becomes important when we look carefully at what Centocor said in the specification, and those are antibodies that have parts of mice and parts of humans, but the human portion is a very small portion at the complimentary determining region. And, finally, the last stage in development was the fully human antibody created by recombinant techniques.

The first fully human antibody that the FDA approved for the treatment of humans was Humira. I think Ms. Elderkin fairly stated what our dispute is. Our dispute is do these four claim terms and the claims that have those claim terms cover fully human antibodies as Centocor contends or does it require that there be some element of something other than a human in the antibody as we contend? Stated differently, are the claims limited to chimeric and humanized antibodies or do they cover fully human antibodies?

Now, I think I can capture the core dispute between us both legally and factually in the next four

or five slides, and I've moved to Slide 7. Ms. Elderkin proposed that the concept of disavowal was an extraordinary circumstance. We know from the many Markman hearings you've had, the Court's very familiar with the basic Markman principles. I just want to address two. One is the concept of disavowal, and the second is concept of the importance of the specification post-Phillips to the disclosed and only embodiment in this case.

And let me say just these three things, Your Honor, the extraordinary circumstance concept of disavowal was a pre-Phillips concept. There was a case -- Your Honor will remember Texas Digital, which suggested broad plain meaning, and out of Texas Digital came two things, one, a lot of controversy, and, two, some cases have said, well, I'm going to follow Texas Digital. The disavowal has to be clear, extraordinary. Phillips has rejected that law and articulated a protocol which Your Honor is very familiar with, but it's made disavowal something less than extraordinary, and it's made the specification more important as it was with Vitronics.

And just to emphasize the point, there are three cases we'd like to offer the Court for consideration. One is the Astrazeneca case, which is a

2004 case. The claim term is solubilizer, and the claim term itself was very broad, but the specification said the solubilizer of this invention has specific features, in this case, something called, if I pronounce it correctly, Micelles, M-i-c-e-l-l-e-s, and criticized solubilizers that didn't have that feature. And the Federal Circuit said, hey, look, the specification has to mean something. If you are criticizing something, even if the broad term covers it, you cannot get claim coverage for that which you have criticized.

The Honeywell case, which was two years later, said the same thing. That case, Your Honor, which is at Slide 8 of our presentation, had a claim term electrically conductive fibers. The question was whether that covered carbon fibers. There is no doubt that as a plain meaning general proposition, it would cover carbon fibers, but the specification included a portion that criticized carbon fibers. And the Court said, well, you can't cover that which you have criticized.

The second concept is simply the concept of the importance of the disclosed embodiment, here, the only disclosed embodiment, and how it should be read post-Phillips. This goes directly to the Centocor argument that an antibody is an antibody, and they're

entitled to that coverage.

Most recently, in 2009, the Federal Circuit dealt with a case that had the broad term wound, and it's a broad term. It has a plain meaning, but the Court said, no, the specification describes it more narrowly. It tells you that they're only dealing with certain types of wounds, and as a consequence, that's the only coverage that you're entitled to.

Why does this become -- why do those two proposition, disavowal, not as an extraordinary event, and the importance of the specification, the disclosure to the claim interpretation become important?

Moving to the factual dispute between us. Your Honor, in the patent at Column 1, Line 4 and then Line 24 to 27, there is something that it appears from Ms. Elderkin's argument we have fundamental disagreement on. Centocor argued here this morning that there was a '91 application. It did say things about chimeric antibodies. It did criticize human antibodies, fully human antibodies, but that was taken out of the patent. That's not true by our view, and at Column 1, Line 4 and then down to 24 to 27, what Centocor said was the earlier applications are incorporated by reference. That's at Line 27.

We'd ask Your Honor to compare Centocor's

position on the '91 application with what it says at Slide 30 when it talked about the Harlow reference, and they said that reference is incorporated by reference because of what's said at Column 12, Line 19 to 20. Incorporated by reference has a meaning. It has a meaning in the patent law. It means it's as if that which is being incorporated by reference is set forth in the specification. And it's going to have the same meaning in Column 1, Line 27, as it does in Column 12, Line 19 to 20, and it means it's incorporated by reference.

Now, that creates, I think, even more confusion than if you just read the specification on its own, but I think it demonstrates that our narrow claim interpretation is correct, and I think it's correct, Your Honor, for two reasons. The first is that if we look at what was incorporated by reference, you're going to see that the invention is described as chimeric antibodies and there is an explicit criticism of human. That's one.

The second, I'm going to walk through the portions of the file history that Centocor has -- I'm sorry, portions of the specification that Centocor has cited to Your Honor, and I think I can demonstrate to you that all of them are consistent with the claim

construction we offer.

So let me turn to the first, which is at Slide 11. Slide 11 has a portion of the original application, and this is now incorporated by reference, and the manner in which the invention, the cA2, was described in the original application is very conventional, and it laid out the background of the invention, it laid out the state of the art, and it described mouse antibodies as well established technology.

But what it said was mouse antibodies have a problem, because they're from mice, when you put them into a human being, the human being is going to recognize them as not human and mice, and the human body is going to do things to reject that foreign -- that foreign substance. That's going to limit the effectiveness of the mouse antibody. So they were clear. Here's a problem with the mouse antibodies.

But they had another section, and this section, which is at Slide 12 from the '827 application at Page 9, this is fully incorporated by reference as a result of what's said in the patent. And what they said is, okay, we have a problem with mice. One possibility would be to consider fully human or human antibodies, but human antibodies have problems. The first thing is

you might like to get human antibodies that are generated in the spleen of a human, but while we can -- we can take spleens out of mice and use them for purposes of creating therapeutics, harvesting spleens from human beings is not going to be something that works.

As a consequence, to get it to work with human antibodies, we need to use a virus as part of the problem -- process. This is that Epstein-Barr virus. Well, that creates some problems, too. It may not work, and the second thing is, it's a virus. If you put a virus in, you could be creating a lots of problems. And then it says, but most importantly, anti-TNF alpha is something that occurs in a human being. So having the human body create an antibody is something that's naturally occurring, doesn't happen naturally. This may not work at all.

So they've said -- if you take my chart with four different possibilities, they said, well, we've got mice at the left-hand end, that works, but it's got some limitations because it's all mouse. We have human at the other end, but human doesn't work. The technologies that are available to make it work could inject viruses and other things into the process, and, ultimately, in the end, it probably doesn't work.

So what did they say? They say, well, the solutions in the middle. And, again, this is -- what's on the Slide 13 is taken directly from the application incorporated by reference -- I should say parenthetically. The last slide on Slide 12 is the portion that Ms. Elderkin said had been removed from the '94 application. That's true, it had been physically removed, but then you have the incorporation by reference which brings back which creates at least infusion. We suggest it brings it back and -- set forth there entirely.

So what does the incorporated by reference application say? It said, well, here is the solution. The solution is a chimeric antibody that has a nonhuman portion and a human portion. And chimeric antibody technology such as that used in the present invention bridges the gaps that we've talked about. That's what they invented. That's what they described as the invention.

And if you think about it, Your Honor, the -- having had them describe the left-hand side and the right-hand side of our antibody slide and say we're in the middle makes some sense.

Now, after that '91 application, there is a really complicated and confusing series of applications

which carry us right up to the point where we have the patents that we have today. And while we agree with Your Honor that resolving priority issues today is not the task, I'm actually going to go through the portions of the -- the portions of the specification Centocor relies upon and identify the application from which they came, because if the Court considers the Federal Circuit's decision in the PowerOasis case, there was a claim construction process Judge Barbadoro in New Hampshire identified the portions that he was relying upon for his claim interpretation. That then led to resolution of the issue that Your Honor alluded to further down the road.

So while it -- there was no resolution of the priority date issue, there was some attention given to just where the portions of the spec came from that the Court was relying upon. And that becomes important in this case because of this complicated history. The interesting thing is even after Centocor tried to -- tried to take this chain of applications and move it in the direction of more human still throughout the preferred antibody is a chimeric antibody.

Your Honor, the Court might pause and say, well, wait a minute, if you really had moved the invention from chimeric to humanized to human and you

now had one that was entirely human, if they put human being that would be fine, why would your preferred embodiment be chimeric? It wouldn't be.

And to go back one slide to Slide 13, here is the reason. I think as the Court knows from its other cases and -- and your service on the Federal Circuit, when someone has a series of applications coming off a first one, you're trying to balance two things that are competing considerations. You're trying to keep that early priority date so that you can eliminate prior art, but you're also trying to get a little bit broader and broader coverage.

Well, that's hard to do if you're trying to keep that priority date, and what you see in this series of applications is Centocor trying to just do that dance which is we want to try to keep '91 as a priority date, but we know we need to get more into the specification if we want to try to cover what other folks are doing, and that's where the confusion comes from.

So, Your Honor, on Slide 16 are the nine portions of the specification which Centocor has either cited to you in its briefs or in its claim construction contentions. And I would say this, I think I can demonstrate to Your Honor that the original 1991 application and the original 1992 CIP are talking about

things other than fully human.

I would say fairly also that there's no doubt that in 1994, the patent lawyers drafting that application were trying to put in concepts that might be a little broader. But I think that I can demonstrate to Your Honor that in doing so, in trying to strike this balance of keeping the '91 priority date, they didn't get to fully human. So if I take them in that order, Column 14, Line 12 to 20, which is on Slide 17, which is the first one, refers only to the human constant region. It's not talking about the fully human antibody. It's talking only about a human constant region which would apply to a chimeric antibody, cA2, and I would say in just a second, Your Honor, I think I'll come to the -- Your Honor asked a question to Ms. Elderkin about the definition of a chimeric antibody. It's actually quite important, even though the claim itself doesn't have chimeric because of the manner in which Centocor has defined the term.

Now, this portion refers only to the human H chain and says nothing about whether the antibody is fully human or not. Centocor says, well, there's references in this patent to a human constant region, and you can see that's all human. The answer is that's true, but we're not -- we're not construing the word

human in the abstract. We're construing the word human as part of phrases. And a human constant region, we all agree, is going to be fully human. A human variable region we actually all agree could be fully human or could not. And so the argument that was made with that phrase doesn't really answer the question, and the fact that this refers to a human constant region doesn't tell you anything about the scope of the claim.

The second portion that Centocor relies upon is Column 19, Line 1-8, and Column 19, Line 17 to 27. This is exactly the same, Your Honor. This is the human constant region which is human and says nothing about the variable region. And if I were to pause just for a second on these two, that makes perfect sense. As Ms. Elderkin described the invention, the invention was a chimeric antibody, cA2, that had a human constant region. The fact that the specification would describe that is completely commonsensical.

The next portion that Centocor relies upon from the original application is at Page -- Slide 19, and it's Column 14, Line 64, to Column 15, Line 9. This says nothing, Your Honor, about whether the antibody is fully human or not. It's referring to a chimeric antibody of the present invention. And then the portion that they cite is talking about transformation of a

human cell. That's -- the portion they're relying upon is that line at Line 69 we just talked about, a process of transformation, not about a fully human antibody. So those are the three that they rely upon from '91.

Then if you get to '92, the concept of human-human is to be sure introduced, and it's introduced in the '92 application. And on Slide 20, we've identified the three places they've identified references to human-human. But this is where Your Honor's question becomes important about the definition of a chimeric antibody, because if I move to Slide 21, which has quotations from Column 20, Lines 45 to 48, and Column 10, Lines 64 to 67.

What the patent says in 1992 is a chimeric antibody, such as mouse-human or human-human. So they themselves in the patent have said a human-human antibody, which our expert has said is not a phrase that scientists use in the normal course, there are other ways to describe it, such as humanized, this phrase that was coined and introduced in 1992, Centocor says this human-human antibody is a chimeric antibody.

Then if you go down to I think the portion of the specification that Your Honor was discussing with Ms. Elderkin, which is definition of chimeric antibody or one definition, chimeric antibodies are molecules

different portions of which are derived from different animal species. So if we put these two things together, the bottom portion from Column 10 is an accurate definition of a chimeric antibody. It's particularly accurate given the invention as Ms. Elderkin described it as cA2, and then they go on 10 columns later and say, okay, now we're introducing the concept of human-human. A human-human antibody is a chimeric antibody.

That takes us to 1994, and on Slide 23 -- Slide 22, we have just put a quotation from the PowerOasis case because it's analogous. Now, to be sure, Your Honor, the decision that was on appeal and affirmed was one that is the event you described as further down the road, but the decision is instructive in that the manner in which the Court identified the portions of the spec it relied upon became important to that decision down the road, and we think that's important because if you move to the '94 spec, if you move to the specification which they rely upon, the real question is are the things that they added enough to broaden the concept of fully human?

We actually still say no if you read it in the full context, but if it does and that's what drives the result, it could become important down the road.

Now, Column 5, Line 55 to 59, does inject

the word human antibodies. If the Court compares the original summary of the invention from the incorporated by reference application and this summary of invention that inserted this word human antibodies, it's like they're from two different patents, literally like they're from two different patents, but the incorporation by reference we say give us -- gives us the answer.

So now we have the word human antibodies. The question is not just what is the ordinary meaning. The question is what is a human antibody as these folks have used it over a period of three, four, five years before the patent office. And the answer can be found in the file history.

On Page 24, Your Honor, we have the original claims of the application that led to the '775 patent. Dependent Claim 3 claims an antibody that has at least one human light chain and one human heavy chain, that dependent claim is dependent on Claim 1, which claims a human anti-TNF alpha antibody. So we have Claim 1 that says we're claiming a human anti-TNF antibody. We have Claim 3 that says that human antibody has at least one human light chain and one human heavy chain, but then when you go to Claim 6, Claim 6 says the light chain and the heavy chain can contain complimentary determining

regions from A2 or cA2.

What does that mean? Well, A2 is a murine antibody disclosed. CA2 is a chimeric antibody disclosed. Claim 6 is telling us that the manner in which these folks have used human antibodies includes antibodies that have mouse at the end. It may be different portions of mouse, but mouse at the end. It's completely consistent with what the invention was and completely consistent with the concept of a humanized antibody.

In Dr. Mark's declaration, he reviewed with the Court some of the different scientific references that use the human antibodies to refer to the humanized antibodies that had been discovered, researched, and reported on in the 1990s. So if we take their first 1994 portion of the spec, human, we look at their very claims, we can see that they're talking about something that is humanized.

Let me take the second, this is the second part of the '94 application, which is at Column 10, Lines 32 to 41. I think probably the most important thing here is, Your Honor, we're not quite sure what the point is. There's nothing about human antibodies. The word human doesn't appear in this portion of the spec. This tells you nothing differently about what the patent

covers.

And then the last portion they rely upon is at Column 18, Line 53 to 62, and I think, Your Honor, this demonstrates the tension that arises when you're trying to keep your priority date and add some material. They rely upon something that says human anti-TNF variable region which contains a framework residue having complimentary determining residues which are responsible for antigen binding. It makes no scientific sense.

When I look -- when I went quickly through our tutorial slide, I said the one point we'd like to make is that that variable region has two parts, the framework region and the CDR. Those are two separate and distinct parts. And their expert, Dr. Adams, said they are distinct. So the portion of the spec that Centocor relies upon actually makes no scientific sense.

So, Your Honor, what that leaves us on this first primary dispute is this, it leaves us with one single disclosed embodiment. It leaves us with an early -- early applications that are incorporated by reference that criticize fully human antibodies. It leaves us with later applications that put the word human in, but in the form of human-human or human, which is described in the claims as including humanized

antibodies.

Against that background, if you look at the full scope of what's disclosed and we consider the public notice function of the claims, the fair interpretation of these claims, we would suggest, the correct interpretation is something that covers chimeric, humanized, but does not cover fully human.

Now, the second issue, whether the phrase competitively inhibits binding of A2 to human TNF alpha requires antibodies to bind to the same epitope of TNF alpha at a defined level of inhibition -- you know, fundamentally, Your Honor, this is a question of are we just going to let the jury sort of try to figure this out on their own or are we going to give this some definition.

And as I sort of struggled with this as we prepared the argument, I thought of a claim that assumed that -- say Ms. Elderkin had invented the jet airplane but decided to claim the jet airplane as a plane with a jet engine that goes fast or that goes faster. That's actually what we're dealing with here is it competitively inhibits. The question is how and to what extent, and what we're urging is that we give the jury some definition of just how and to what extent.

On the screen now on Slide 30 is our claim

interpretation, and, actually, if you take them in reverse order, if you take the second portion, we are describing the cause. We're describing the fact that it's the binding to the same epitope that causes the inhibition. Then we're saying in the first paragraph, and here's the effect. Here's how much. Here's how much faster it has to be. It needs to be at least as well. Now, parenthetically, at the very end on the Slide 30, we have a sentence that says an epitope consists of Amino acid residues on the antigen to which an antibody binds. Centocor suggested that that was inconsistent with what's in the specification. We would just ask the Court to consider the words because it's not inconsistent at all.

On Slide 31 is Centocor's definition, which we would suggest fundamentally just tosses the issue up in the air to the jury with no guidance. So if we take our construction in the order that I've suggested, which is cause and then effect, the bottom portion deals with cause and the causes binding the same epitope of TNF alpha.

As the Court knows from the tutorial, two antibodies can inhibit each other in a number of different ways. They can bind to the same epitope in this overly simplified version, and that would be, in

our view, competitive inhibition, but also one epitope can bind to a site and block the other from getting in, and that's called steric hindrance.

On Slide 34, another way that inhibition can occur is if one binds and when it binds it causes a cell to change its shape so that the antibody can't get to the antigen binding site or to the epitope binding site, and that's called allosteric hindrance. And the last, which may be the most predictable is the antibodies just bind to themselves and make themselves useless.

Which of these different things is the patent talking about? And, again, the answer is in the words of the specification. In the specification itself, they say Figure 9A and 9B, which Ms. Elderkin put up on the screen, is an example of a cross blocking epitope. And they say -- I'm trying to eliminate that red arrow, but I can't. You did. Thank you. They say themselves that this cross blocking epitope is what they're talking about as competitive inhibition, competing for the same site is what they're talking about.

And the patent at Column 13, Line 15 to 26, defines what the epitope is. So we suggest it's pretty clear, but if the Court was concerned and thought that there was ambiguity in what they said themselves, the

file history answers the question by saying, thus, the same -- claimed monoclonal antibodies, in their ability to inhibit A2 binding, must also bind to the same or similar epitope.

Now, we also suggest in addition to cause, there has to be some way to quantify the effect, and I think this becomes important not just in the abstract, Your Honor, because the Federal Circuit says we ought to tell the public when their jet airplane is going fast enough to be fast or fast enough to be faster. We need to tell them what the scope of the right to exclude is. I'm going to come back to this one.

A key here is that Centocor's expert gave testimony during the Markman depositions which demonstrates that just the phrase itself, competitively inhibits, doesn't tell you a whole lot. He said, well, you know, it's sort of like the United States election. If it's a landslide, you know it's a landslide. If it's close and the balance is in Florida, then it's close.

I can tell you on each extreme what it is, but I can't tell you what it is in the middle. He said it's a matter of degrees. 5 percent would be enough -- would not be enough. 65 percent, 75 percent, 35 percent, I don't know. And we suggest that that would create an indefiniteness problem.

Now, Ms. Elderkin said that indefiniteness is not something the Court considers at Markman. I would suggest that also is old law. As Markman came into being, it was this old law that said indefiniteness was a question of fact. Markman became a question of law. Post-Markman and post-Phillips, the question of whether something is definite has to be become a legal issue for Your Honor. And if it is indefinite, it is indefinite. If it's capable of being given a definition, it's capable of being given a definition.

And all we've tried to do is say, okay, if it's going to be given a definition, let's give it a definition. And that's why we explained to Your Honor in the tutorial the cross blocking epitope test, the need for a positive control, the need for a negative control, and then the test antibody. And if the Court considers Slide 39, what ought to happen, if you're binding to the same or similar epitope, the positive control, which is our green line, when you put the test body in, it ought to get pretty close to the same, and that is what the patentee was talking about, because if we move to Slide 43, at Column 12, Line 4 to 15, they say, it's having substantially the same specific binding site.

Now, Your Honor, very quickly, let me say

this on the four remaining terms. On recombinant or produced recombinantly, having heard the argument, I'm not sure that there's a difference more than words, and if -- you can take something off the Court's plate if what Ms. Elderkin represented to be their definition of recombinant and produced recombinantly means that claim interpretation is fine with us.

As to specificity, as I think we said in our brief, Example 10 does describe both. We think probably the right descrip -- the right definition includes both.

And then as to inhibits pathological activity, I'm going to leave it to the briefs.

But the final one I'm going to talk about is this binding to a neutralizing epitope of human TNF alpha in vivo. Your Honor, this is one where I think looking at this, looking at what occurred answers the question. The asserted claims of the '775 patent include this claim limitation with the word in vivo. In vivo means in a living thing as we've suggested. The '239 patent, which came out second and the second patent before Your Honor, eliminated that claim term.

Now, if the definition is as broad as in vivo, which is a series of calculations that are made outside of all living things, if it's as broad as Centocor says, why bother to take the claim term out in

the '239 patent? The fact they took the claim term out I think answers the question. The word in vivo is there. The word in vivo has a meaning. It is a test that measures something in a living organism, and to the extent that Centocor's argument is you can't do this measurement in a living organism, our answer is three things.

One is, well, if you can't, you shouldn't have put it in your claim term, and Chef America says we take it isbut. The second is it's in the claim term, and it's a requirement, and if Abbott doesn't do it, Abbott doesn't do it, or if you can't prove it, you can't prove it. And, lastly, the fact that you took it out in the '239 patent is the answer to the question of whether the interpretation is broad as we suggest today.

So with that, Your Honor, unless there are any questions, we would rest on the briefs as to the remaining issues.

THE COURT: I don't believe I have any. Thank you, Mr. Lee.

MR. LEE: Thank you, Your Honor.

THE COURT: Rebuttal?

MS. ELDERKIN: Just a few comments, Your Honor.

THE COURT: Yes.

MS. ELDERKIN: Mr. Lee pointed to a slide where we had the claims that were originally presented in the '775 patent application, and those claims did say human antibody. I think the point that we'd like to make clear is that the term human antibody -- again, as the term human is construed by Centocor, and we think properly, human means derived from human DNA, but human just as a human antibody can include a humanized antibody. The term human antibody as used in that claim wasn't limited to a fully human antibody. So the claim didn't say fully human antibody. It said a human antibody. So it was not inconsistent that there was a subclaim that referred to there being CDR regions that were of nonhuman origin.

And I guess the only other thing I'd like to mention is Mr. Lee used the example of the jet engine and how the competitive inhibition would be unclear because we don't know if it's faster-faster like the jet engine. And I would submit that that's really not the proper analogy.

Both experts have testified in their depositions of excerpts have been provided in the briefs that those skilled in the art know from the result of a competition assay whether there's competition or not, and what Abbott is trying to do is to incorporate into

the claim the reason for the competition or the reason the jet engine is going faster, not that it is going faster or not, and that's not -- that's an improper thing to do with these particular claims.

Those skilled in the art would know when there's competition, and if they wanted to quantify it so that they compare one antibody to another, they could do so by mechanism by assays described in the Harlow reference, but it's not necessary to do so to know whether two antibodies compete. In fact, Dr. Marks, Abbott's expert, well, you look for a trend. If you don't see any competition at 15 percent or 5 percent, we look for a trend. We add more antibody and see if there's a trend. So it's not necessary to quantify it to know whether there's competition.

That's all I have.

THE COURT: What do you say about Mr. Lee's argument -- he cited me, I think, three or four cases about you can't criticize something in your -- and then claim it, I believe was basically what he was arguing to me about a couple of cases or three cases, and he's saying that in your original application, which you have reincorporated by reference, what do you say about that?

MS. ELDERKIN: Okay. A couple of things. First, I didn't mean to imply that we disagree that it's

incorporated by reference. It certainly is. It's just the text that was in the original application no longer appears in the patent, and I think there is relevance to that. I'm not aware of any cases that have ever addressed this issue.

THE COURT: I'm not either. I was hoping somebody was going to cite me one that said that exact thing. He did cite me three cases, I believe, that talked about, though, that you can't criticize something and then claim that which you have criticized was, I believe, what the principle was that he was arguing to me.

MS. ELDERKIN: There are cases about that, and I think that those particular cases are very different from what we have here. The criticism, so to speak, if you want to call it that, of human antibodies that appeared in the '827 application, the original application, was not that human antibodies are bad or we can't have human antibodies. That certainly wouldn't be part of our invention. It really wasn't criticism. It was saying, gee, it would be difficult to make these using the technology that Mr. Lee referred to, or you have to use a virus in a human and everything.

But elsewhere in the -- in the '827 spec, and I referred to this in my slides this morning, and,

I'm sorry, I don't have it in the top of my mind, elsewhere in the spec there is a disclosure of a way that one could use to make human antibodies. It's -- it's in there in the original one.

So the -- we would disagree that it was criticism of human antibodies in the original specification. It was a reference to the fact that, gee, these would be hard to make, and there was another way to make them, and then, of course, when the application was refiled, there was even more information provided of all that of what was already known in the art.

I think the other thing that we would have to point out about this incorporation by reference, I agree it's incorporated by reference, but I think if we took everything that was incorporated by reference and laid it all out along with everything that's actually in the printed patent now, it would not lead to a clear conclusion that there was criticism and that anything was disavowed, because even if there were some criticism of human antibodies, and, again, we don't say that there was, elsewhere, there's reference to the human-human antibodies and to human antibodies. And looking at it all in total, one skilled in the art wouldn't walk away from that and say, oh, they've criticized human

antibodies, so this patent certainly can't encompass them.

THE COURT: Thank you.

MS. ELDERKIN: Thank you.

THE COURT: All right. I should -- this case is set in -- for jury selection on May the 29th, which is a Friday. We put this on -- jury selection on the 29th because counsel on one side or the other had some problems the first part of June, and I gave you a firm trial setting that we would go to trial on the week of the 15th.

That's before the Court had a judicial conference committee that's been scheduled for the week of the 15th. So the earliest you'll go to trial is June the 22nd. I just want -- I know y'all -- I just hadn't picked up I had it specially set for the 15th, and I also had this later set matter that I had -- since those committees are reported by the Chief Justice, I try to attend those committees.

Yes?

MR. SAYLES: May it please the Court --

THE COURT: Now, wait a minute. Have you got another problem you want to tell me about?

MR. SALES: No.

THE COURT: I want to talk to you about

chimeric antibodies.

MR. SAYLES: Well, Judge, now, my job is to translate that into East Texas English language.

THE COURT: Well, let's -- you want to talk about that today, or you're not prepared?

MR. SAYLES: I'm working on that.

THE COURT: Okay. All right. What do you want to talk about, Mr. Sayles?

MR. SAYLES: Judge, in this case, as the Court knows from the file, we're not seeking an injunction, and I wanted to inquire if we can get some guidance on whether we would be expected to present the future damages to the jury or whether that will be a matter that will be handled depending on the verdict and post verdict.

THE COURT: Well, Judge Folsom is -- which case did he have where he tried to set the future damages and he did it wrong or something? What we have done in the past two cases is not take on the future damage questions just until the time of trial, and then try the future -- try them -- once we got the royalty rate, then we try them later.

MR. SAYLES: All right.

THE COURT: We direct -- at the time I enter a judgment, I generally direct the filing of a second

case, sever that.

MR. SAYLES: We knew that was the way that it had been done.

THE COURT: If y'all have got something better --

MR. SAYLES: No.

THE COURT: You know, I'm not wanting to try the case the second time. Don't get me wrong. I'm not looking for another case. I'm just -- that's what Judge Davis has done, I believe, and Judge Folsom has -- and I haven't gotten any clear direction from the Circuit to --

MR. SAYLES: And that's why I inquired. No, with 12 and a half hours a side to try a case involving recombinant chimeric antibodies --

THE COURT: Have I already set your hours per side?

MR. SAYLES: You did say 12 and a half, but we'll --

THE COURT: Why did I give you that many? I mean, since like I gave you an hour and a half -- were you saying that was too much Mr. Sayles?

MR. SAYLES: No, sir. I was not. I was saying that the way you normally handle future damages will be just fine.

THE COURT: Well, we can -- I mean, you know, if y'all think you're going to really need more than 12 and a half, you're going to need to tell me why.

MR. SAYLES: Yes, sir.

THE COURT: If you can't do it in -- you know, if you really can't, but up until now, I have yet to have anybody run out of time. The only case that came close, somebody actually went over about a minute and a half, but it was an antitrust case. It was not a patent case.

MR. SAYLES: Well, we're planning 12 and a half, Judge.

THE COURT: What do you think about that, Mr. Lee?

MR. LEE: I think we should be able to do that, Your Honor.

THE COURT: Okay. Agreement. I'm out of here.

COURT SECURITY OFFICER: All rise.

(Hearing concluded.)

CERTIFICATION

I HEREBY CERTIFY that the foregoing is a true and correct transcript from the stenographic notes of the proceedings in the above-entitled matter to the best of my ability.

SHELLY HOLMES Date
Deputy Official Reporter
State of Texas No.: 7804
Expiration Date: 12/31/10